PERINATAL OUTCOME IN HYPERTENSIVE DISORDERS OF PREGNANCY TREATED AND UNTREATED CASES

THESIS

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C E R T I F I C A T E

This is to certify that the work entitled " Perinatal outcome in hypertensive disorders of pregnancy, treated and untreated cases ". which is being submitted as a thesis for M.S.(Obestetrics and Gynaecology), has been carried out by Dr. Upma Gupta in the department of Obstetrics and Gynaecology, M.L.B. Medical College, Jhansi. The candidate has fulfilled the necessary stay in the department as per University regulations.

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CONTENTS

Page

1. INTRODUCTION ... ... 1-3 ...
2. REVIEW OF LITERATURE. ... 4-28
3. MATERIAL AND METHODS. ... 29-40
4. OBSERVATIONS 41-57
5. DISCUSSIONS 58-64
6. SUMMARY AND CONCLUSIONS... 65-66
7. BIBLIOGRAPHY 67-74
8. APPENDIX ... ...

INTRODUCTION

INTRODUCTION

Hypertensive disorders of pregnancy (HDP), regardless of underlying etiology and pathogenesis has a wide spectrum of clinical manifestations and are encountered by every obstetrician because of it's high incidence.

In developing countries hypertensive disorders of pregnancy accounts for a sizable numbers of perinatal deaths, due to lack of facilities for careful foetal monitoring.

Although in few epidemiological surveys from teaching institutions in India, considerable attention has been paid to maternal and perinatal mortality in eclampsia, only anecdotal information exists with regard to mild to moderate preeclamptic toxaemia. This apparent lack of interest in mild cases of toxaemia may not be justified because 60-75% of cases of toxaemia are of mild to moderate variety.

This study reviews our experience of perinatal outcome in patients with hypertensive disorders of pregnancy with comparison of results in every type of hypertensive disorders of pregnancy right from the pregnancy induced hypertension to the eclampsia.

and usually better than, in the untreated women. The wide assay of effective drugs available to treat hypertension allows on case of blood pressure control not attenable in the past.

In this study we also tried to evaluate the various casual and contributory factors or perinatal mortality, the results of which may suggest measures to reduce perinatal mortality rate in hypertensive disorders of pregnancy.

The influence of pathophysiologic changes during pregnancy can, perhaps be judged best, by their effect on the foetus, upon such events as the occurance of intrauterine growth retardation, neonatal deaths and neonatal morbidity, collectively this what is meant by the outcome of pregnancy.

Our second aim of study was to observe the effects of antihypertensive treatment on the perinatal outcome. The encouragement in this direction was obtained from the good results seen in the nonpregnant hypertensive population, in which the vascular complications of hypertension can be prevented with the antihypertensive therapy.

In pregnant hypertensive patients, the approach to therapy varies with the circumstances but there is now unequivocal evidence that treatment of the hypertension does not worsens and in most instances improves foetal survival. The extraordinary advances in the treatment of hypertension in the nonpregnant population have completely altered the prognosis of hypertension and vascular disease and there is no evidence that the treatment of hypertension during pregnancy compromises foetal survival. In every instance in which careful observations have been made, foetal survival in the treated group is at least as good as,

REVIEW OF LITRATURE

REVIEW OF LITERATURE

Hypertension during pregnancy causes appreciable increase in maternal and fetal morbidity and mortality. Pregnancy associated hypertension tends to be more important in this regard than essential hypertension, particularly when severe or accompanied by proteinuria. Current knowledge allows a rational scientifically based approach to the clinical management of hypertensive pregnant women. The trial results are strong and consistent and support the active treatment of hypertension during pregnancy.

Blood Pressure during pregnancy

Arterial blood pressure is a product of cardiac output and peripheral resistance. The cardiac output is measured basing upon cardiac index. This is expressed in terms of litera/minute/sq.mt. of body surface area. Naturally this is dependent upon the circulating blood volume and stroke volume. Increasing stroke volume is major component responsible for greater cardiac output in pregnancy. In most women both the systolic and diastolic blood pressures fall a little in the first 6 months of pregnancy, then rises to pre-pregnancy levels before term (Mac Gillivary et al 1969).

Factors influencing blood pressure

Arterial pressure varies over a wide range in normal non pregnant individuals and during a 24 hours period of continuous recording, the highest levels are often twice the lowest (Beren et al 1969). Large changes occur during excercise, conversation, mental rithmetic, dacfecation, Copulation and sleep (Beren et al 1969) and progressive lower values are found as the subject becomes familiar with her surroundings (Armitage and Rose 1966). Blood Pressure varies widely in different communities on even in different parts of the same community (Hawthorne et al 1969), and it may be associated with increased dietry sodium and obesity (Evans and Rose 1971). This is well known that blood pressure tends to increase with age (Pickering 1968) woman with pregnancy induced hypertension are more likely to have female relatives with a similar condition then are women with normal blood pressure during pregnancy (Adams and Finlayson 1961), although other studies have not confirmed this (Selan et al 1970).

Generally, nonpregnant females appears to be at less risk than males for any given age and blood pressure (Pickening 1968), but this is not the case if the woman is pregnant. Pickening (1968) has shown that the development of accelerated (malignant) phase

hypertension, with its complications of acute left ventricular failure, hypertensive encephalopathy and cerebral haemorrhage, is associated with very high levels of blood pressure (diastolic usually greater than 130 mm Hg) in the nonpregnant subjects, while in pregnancy, these complications may occur at a much lower diastolic pressure (i.e. 110 to 120 mm Hg) and eclampsia can accompany diastolic pressure of 90 to 110 mm Hg. The response of the blood pressure to pregnancy and the critical level for the development of complications appear to vary the different parts of the world (Davis 1971).

Pathogenesis of pregnancy induced hypertension

The cause of preeclampsia is not known but a number of deductions can be made from the available evidence. The presence of trophablast is essential while a fetus is not, as preeclampsia can develop with hydatiform mole (Chun et al 1964). Preeclampsia must therefore be associated with either an abnormality of trophoblast itself or of the maternal adaptation to the presence of trophoblast, the the latter is more likely as there are a number of maternal specific risk factors for the development of preeclampsia such as primigravida (Mac Gillivary 1958), family history (Cheeley 1980) and underlying medical disorders (Pelding 1969). Fetal or paternal factors have

been implicated (Reed et al 1985), but the evidence is circumstantial.

A normal pregnancy is associated with many changes in maternal physiology to result largely from humoral factors which enter the maternal circulation from the placenta and amnion chorion. It is most likely that the various manifestations of preeclampsia are similarily mediated. If they are caused by the presence of some abnormal circulating factor, or the absence of some normal factors, then susceptibility of the target organ system becomes a critical factor and this may explain why women with chronic hypertension (Walters 1966) or renal disease (Pelding 1969) are more likely to develop preeclampsia.

Placental insufficiency

Placental ischaemia could be the cause of the preeclamptic syndrome (Page 1972). A maternal syndrome resembling preeclampsia can be induced experimentally in baboons (Covarsh et al 1977), dogs (Abitol 1977), rabbits (Abitol et al 1976) or rats (Abidol 1982) by reducing placental perfusion, although some workers have been unable to confirm this (Reddy et al 1984).

Placental insufficeny could occur via a numbers of different mechanisms. The failure of the

normal adaptation of the spiral arteries, in response to trophoblastic invasion may be the cause. In the early stages of a normal pregnancy, proliferating cytotraphablast cells invade the spiral arteries in a retrograde direction as far as the radial arteries, deep in the myometrium, the muscular and elastic tissue of the media is destroyed, and replaced by fibrinoid material so that the spiral arteries of the placentral bed becomes dilated and funnel shaped (Brosens 1977).

In pregnancies, complications by pre-eclampsia these physiological changes are confined to the decidual, segment of the spiral arteries and denot reach the myometrial trunks (Brosens et al 1972, Gerretsen et al, 1981, Hustin et al 1983). Thus, there is poor placentation, particularly affecting the maternal blood supply. This decrease in uteraplacental blood is responsible for intrauterine growth retardation and the more severe the preeclampsia, the greater the comprise (Lunell et al 1982a).

In other situations, an excessive placental mass or sclerotic uterine vessels could result in placental ischaemia.

<u>Immunological factors in hypertensive disorders of pregnancy</u>

Failure of normal traphoblast invasion of the

spiral arteries could be due to immunological factors, the hypothesis is that, the maternal immune response to trophoblast needs to be down regulated to permit normal invasiveness. The down regulation may depend directly on immune responses generating 'blocking antibodies' or suppressor cells (Redman et al 1984). A number of the epidemiological features have provided circumstantial evidence for the importance of immunological mechanisms, but so far there is little direct evidence. The protective effect of first pregnancy (MacGilli very, 1958) is possible but unconfirmed (Compbell et al 1983), protective effect of previous abortion and of blood transfusion (Fenny et al 1977), the increased incidence in multigravida who change partners (Fenny and Scott, 1980) or have donor insemination pregnancies (need et al, 1983) are fifficult to explain on an immune basis. The assumption to that a beneficial (i.e. immuno regulatory) response to foetal antigens occurs, and is absent in women developing pre-eclampsia (Scott et al 1978).

Change in total peripheral resistance in HDP

Preeclampsia is associated with an increase in the peripheral vascular resistence (Benedeth et al, 1980; Carlsson 1984; Greenediyk et al, 1984), while cardiac output is maintained. An increased cardiac output has been documented by some workers at the time of

delivery or shortly after words (Benedtti et al 1980; Phelon and Yurth 1982; Henderson et al 1984; Cotton et al 1984).

Increase in peripheral resistence is central in the problem of hypertension, irrespective of the type of hypertension. The peripheral resistence can be increased due to change in either intimal layer or medial layer of vessels or in both of them.

Alteration in the intimal layer has been reported by Brasens and Benzer 1972 in form of atherosis which in condition of stress is likely to produce vasospasm and rise in blood pressure. Preeclampsia is accompanied by the development of a characteristic glomerular appearance termed 'glomerular capillary endotheliosis (Lindheimer and Kartz 1977).

The smooth muscle of the tunica media responds to neural and humoral factors: Neural influences particularly in pregnancy have not been provide to be very important cause, but certain humoral factors definitely regulate the tone of vessels wall.

a. Catechalemines

b. Angiotensin

c. Nak AT Pase, Calcium

d. Prostaglandins, Thromboxanes and Prostacyclin

e. Clotting system

f. Other circulating factors.

(a). Catecholamines :- Transient labile hypertension in young age is usually considered to be due to the catecholamine effect (Stereo 1977). However in cases of pregnancy induced hypertensive a direct relationship of epirephrine or norephinephrine with the onset, outcome and severity of the disease has not established.

The 'grip test' reported from Israel (Rebinovici et al 1985) indicated the differential response in the second stage of labour with bearing down activity of the patient, between the hypertensive and normotensive pregnancies. In the case of former there is a rise in catecholamines with tachycardia and hypertension. Whereas in normotensive pregnancies tachycardia and hypertension are absent, inspite of increased catecholomines.

Zuspon and Kowada (1976) reported an increased urinary excretion of noradrenaline and adrenaline in hypertensive pregnancies, but Pedersen et al failed to find any change in plasma noradrenaline level in hypertensive pregnancies.

(b). Angiotensin :- Symonder (1981) observed a marked activation of renin-angiotensin system (RAS), with elevated plasma renin activity (PRA), plasma renin concentration (PRC), renin substrate, All (Angiotensin II) and aldosterone in normal pregnancy. In preeclampsia there are

reduced levels of PRA, PRC(Kokot and Cekenski 1972, Vein et al 1973, Pedersen et al 1982b, 1983, Beilin et al 1983, Korlburg, et al 1984 and All (Vein et al 1973, Beilin et al 1983), suggesting that the renin angiotensin system is not of primary importance in the pathogenesis of the disorders. However, elevated PRA (Symonals et al 1975, Annat et al 1981) and All (Symonds et al 1975) have been reported in hypertensive gravidae.Also a positive corelation between All levels and blood pressure in primigravidae, but not multigravidae, has been reported (Symonds and Brough ten Pipkin, 1978) so the picture is not clear.

The Chorio-decidua contains renin substate (Graven et al 1983) and synthesises active and inactive renin (Warren et al 1982). Uterine renin, by control of all and prostaglandin synthesis locally, may be a regulater of uterine blood flow (Ferris 1982). In a small study, peripheral and uterine venous samples were obtained at caesarean section (Braughton Pipkin et al 1981). Peripheral PRC was low in hypertensive subjects, but uterine veins levels were nearly twice as high, whereas there was no gradient in normotensive women. A similar gradient of PRA in peripheral and uterine venous blood has also been found (Kokot and Cekenski, 1972). All levels were higher in the preeclompatic group but

there was not a significant gradient between peripheral and uterine vein levels (Broughton Pipkin et al 1981).

(c) Nak AT Pase and Calcium

Recent studies have thrown considerable light on the mechanism of hypertension in general on the basis of intracellular status of sodium, which is regulated by sodium pump. Under normal circumstances 3 molecules of sodium are egected out of the cell in exchange of 2 molecules of potassium (Aample and Lever (1986). In disorders genetically or otherwise determined there may be faulty function of Na-pump leading to retention of undue amount of sodium inside the cell. It is further established that high concentration of sodium may be responsible for increase in pressor response to calcium ions.

(d) Prostaglandins, thromboxanes and prostacyclin

Prostaglandin E (PGE) is a potent vasodialator (Sperof and Serfman 1977) and plasma level increases in normal pregnancy (Bey & Ferris, 1979). Prostaglandin F (PGF) has vasoconstrictor properties and may decrease uteroplacental blood flow (Pulkkinen et al 1975). Urinary excretion of PGE and PGF, which reflects renal production (Frolich et al 1975) increase in normal pregnancy, but PGE excretion is lower in preeclampsia. While PGF excretion is

unchanged (Rathaus et al 1982, Moutquin & Leblanc 1982, Pederson et al 1983). Reduced PGE and increased TFG, production of placental tissue from preeclamptic women has been reported (Demer & Gabbe 1976).

Prostacyclin, the major prostanoid synthesized in the walls of arteries and veins is a vasodilator which also prevents platlet aggregation, while thromoxane A_2 induces platlet aggregation and constricts arterial smooth muscle (Moncada & Vane, 1979). There is an increase in circulating prostacyclin in normal pregnancy as judged by measurements of circulating 6 Keto PGF 1 alpha (6 Keto prostaglandin F I alpha) (Lewis et al 1980). Conversely a defeciency of prostacyclin production could explain the generalised vasoconstrictior, increased platlet consumption and depressed PRA which are the characteristic of preeclampsia (Lewis 1982).

(e). <u>The clotting system</u>

There can be no doubt that the coagulation changes which occur in pregnancy induced hypertension play a major part in the pathology of the disease. The question that remains unanswered is whether there changes are primary. Abnormal activation of the clotting system occurs in preeclampsia (Bonnar, 1977, Howie, 1977), distinguishing this disorders from essential hypertension (Howie et al 1976). The process proceeds in two stages, first is well compensated, while the second stage is characterized by dissemi-

nated intravascular coagulation (DIC). Although fibrin degradation products (FDP s) are raised in only some cases of preeclampsia (Howie, 1977), there were elevated levels of fibrino-peptide-A (Macouglar et al 1982, Borok et al 1984) and soluble fibrinogen/fibrin complexes (Pckillap et al 1976). Fibrinogen levels are generally normal (Inglis et al), although it may fall slight in severe cases. (Thorburn et al 1982) or may be elevated (Lox et al 1983, Long et al 1984).

The ratio of factor VIII related antigen to factor VIII activit, is elevated in preeclampsia (Thornton or Bonnar 1977, Redman et al 1977), a change which is thought to indicate increased thrombin activity and which may occur in advance of other clinical manifestations (Redmon et al 1977a). A slight, but significant fall in antithrombin III levels has been reported (Weenik et al 1983) and corelates with the severity of the disease (weenik et al 1984).

A reduction in platlet count is common in preeclampsia (Gules & Inglis, 1981) and often develops early in the course of the disease (Redman et al 1978). Platlet life span is shortened (Rakoczi et al 1979, Inglis et al 1982), levels of plasma beta thromboglabulin are raised (Inglis et al 1982, Douglas et al 1982) and abnormall large circulating platelets, reflecting a young platelet

population (Giles & Inglis, 1981) are seen.

(f) Other circulating factors

Other mediators of vasoconstriction, possibly released from the utero-placental circulation have been sought, but their demonstration is doubtful (Chesley, 1978c). A recent report of a heat labile vasoactive substance in sera from preeclamptic women (Fuller et al 1982) included only four controls and the significance of the observations can not be assessed. A circulating digoxin-like substance has been detected in preeclampsia (Gusdon et al 1984, Graves & Williams 1984), but its role is uncertain.

MATERNAL AND FETAL COMPLICATION IN HDP

Maternal complications

Short term complications : These are uncommon, but potentially lethal, especially if several complications arise in the same individual.

1. Eclampsia
2. Cerebrovascular accidents, usually intracerebral haemorrhage, occasionally ruptured intracranial aneurysm or cerebral thrombosis. This may be fatal or the women may be left with a residual neurological defect such as hemiplegia, dysphasia or visual distrubance.
3. Accidental haemorrhage.
4. Acute left ventricular failure with pulmonary oedema.
5. Acute renal failure.

6. Micro-angiopathic haemolytic anaemia (Haemaltic uraemia syndrome).
7. Disseminated intra vascular coagulation.
8. Hepatic failure.
9. Side effects of drug therapy.

Long term complications

The occurance of eclampsia does not influence the long term outcome of pre-existing hypertension (Chesley et al 1968). The evidence to date suggests that hypertension and proteinuria induced solely by pregnancy, with no other apparent cause, donot appear to predispose to subsained hypertension or renal impairment in later life (Adams and Mac Gillivary 1961, Chesley and Platlet 1959, Epstein 1964). However pregnancy in some women may unmask a latent genetic predisposition to hypertension which settles after delivery, but recurs permantly later. Such women usually have a strong family history of raised blood pressure (Adams and Mac Gillivary 1961).

Fetal complications

1. Intra uterine death.
2. Poor intrauterine growth - associated with "Placental ischaemia".
3. Neither or these complications appears to be related to the actual level of blood pressure, of more importance is the duration of the hypertension and the presence of proteinuria (Leather et al 1968, Walters 1966).
3. Immatunity and prematurity with associated hazards of

neonatal death and pulmonary renal and hepatic dysfunction.

4. Brain damage e.g. cerebral palsy. This is likely to be due to anoxia associated with (a) poor placental blood flow (b) maternal hypoxia induced by eclampsia or excessive therapy with sedatives and anticonvulsants.

5. Side effect of hypertensive drugs e.g. intestinal ileus, thrombocytopenia and pancreatitis.

Role of Antihypertensive treatment

There is still controversy about the need to treat mild hypertension in non-pregnant subjects, particularly when the diastolic pressure is below 100 mm Hg (Ferris 1982, Lancet 1980), the aim of treatment being to prevent long term cardiovascular complications. There is little evidence to justify treatment of mild to moderate hypertension in pregnancy on grounds of maternal welfare and the cause for treatment depends on influencing pregnancy outcome favourably. Treatment has been advocated to prevent development of pre-eclampsia in woman with essential hypertension (Ferris 1984) and to prevent the progression of mild pre-eclampsia, once it is established (Luble, 1984).

To have a beneficial effect the underlying process, the preeclamptic lesions of the spiral arteries would have to be prevented. As both restriction of physiological changes and acute atherosis occur in normotensive pregnancies with growth retardation, it seems unlikely

that either they result from hypotensive injury or that they could be prevented by antihypertensive treatment.

There are clear maternal indications for controlling severe hypertension. A blood pressure of 170-180/110-120 represents mean arterial pressures of 130-140 mm Hg, close to the limits, beyong which experimental vascular damage begins (Goldby & Beilin, 1972). An acute reduction in blood pressure could be expected to reduce utero-placental blood flow, but if vasospasm is a significant factor, vasodilatation could off set the effect of pressure reduction. Acute reduction of blood pressure with hydralazine from severly hypertensive to normotensive levels has been associated with fetal heartrate deceleration in some patients (Vink et al 1980), and decreased placental blood flow has been inferred from reduced metabolic clearance rate of dehydroisoandrosterone sulphate (Gant et al 1976), while more direct measurements have shown a reduction marked in some individuals, but not significant overall (Iouppileet al 1985). Reduction of moderately elevated blood pressure to normal with labetalol or hydralazine does not alter utero-placental blood flow (Zuneli et al 1982b, 1983).

Methyldopa as antihypertensive drug

Methyldopa acts centrally and possibly peripherally, decreasing blood pressure by a fall in peripheral resistence, then in cardiac output (Bochner et al 1983). With high dose treatment, sedation is very common in the

first 24 hours, and transient oliguria is also frequent, but not a cause for concern if blood urea and creatinine are normal (Redman 1977). Depression is a recognised side effect of treatment with methyldopa. Although in a trial of its use in pregnancy depression was no more frequent in treated women than controls (Redman et al 1976b).

Methyldopa remains our drug of choice because of the wide experience of its use in pregnancy and the reassuming follow up of 100 children exposed to the drug in utero, at 1 , 4 and 7 years of age (Mutch et al 1977, Gunsted et al 1980, Cockburn et al 1982). The average head circumference of the neonates was slightly but significantly smaller in treated cases (Moor et al 1978). The neonates involved were born to mothers who started treatment between 16 and 20 weeks gestation and it may be a chance finding. No effect of methyldopa on head circumference was seen in a other smaller trial (Fidler et al 1983).

Role of Diuretics

Diuretics lower blood pressure and reduce the oedema but donot significantly reduces the incidence of proteinuric preeclampsia or improve perinatal outcome (Redman 1984, Collon et al 1985) and are not useful in established preeclampsia. Serious side effects, although rate (Collin et al, 1985), have occured. They cause hyperuricaemia (Landersman et al 1985). Obscuring a useful sign, may aggrevate hypovalaemia (Sibai et al 1984) and reduce

placental perfusion and metabolism (Gante et al 1975).

Severe pre-eclampsia is associated with an increased incidence of perinated loss and IUGR (Chamberlain et al 1970 Im et al 1982, Brazy et al 1982, Sagen et al, 1982), particularly if it presents early (Moore & Redman 1983), leading to iatrogenic preterm delivery (Benedetti et al 1982, Sibai et al 1984.

Gibson et al (1950) studied immediate prognosis in cases of toxaemia in late pregnancy. The authors reported the incidence of maternal mortality in 0.28% cases and foetal salvage in 11.7% cases. In this series half of the still births were macerated. They also observed a higher caesarean section rate in the toxaemic group in comparison to the average hospital caesarean rate. These workers found that the conservative obstetrical treatment is useful only before 35 weeks of pregnancy and an early delivery gives better foetal results after foetal maturity.

Walters et al (1960) observed the effects of sustained maternal hypertension on foetal growth and survival and found the perinatal mortality rate to be 72.7% per 1000 total births in comparison to 26.5 control group. They observed that the incidence of prematurity war 16.3% and the incidence of foetal distress was 29%. The authors reports 32.7% H.D.P. cases from the elderly age group (more than 30 years). In this series, no effect of blood pressure was reported on the foetal birth weight.

Mac Gillivary et al (1961) in their study corelated occurance of perinatal mortality and prematurity with the severity of the blood preasure and albuminuria. They concluded that those women were more prone for recurrence of toxaemia, who had such history in previous pregnancy Leather et al (1964) carried out a controlled trial of hypotensive agents in hypertension in pregnancy. They observed that antihypertensive agents were helpful in cases developing before 20 weeks of pregnancy, to improve the outcome of pregnancy, while these were of little help in cases developing toxaemia in later part of pregnancy, especially along with proteinuria.

Smith et al (1966) reported their experience with the use of methyldopa in the management of severe hypertension in pregnancy & they reported the perinatal mortality in 6.2% of study cases.

Hendrieks et al (1968) in their study of a group of toxaemic patients, evaluated the relationship between fetal weight, fetal survival and the maternal state. The authors concluded that birth weight of babies of toxaemic mothers was lower than of control group, and premature deliveries were more common in toxaemic group. They observed that the perinatal mortality was twice in comparasion to the control group. In this series incidence of preeclampsia was three fold higher among primigravida than among multigravidas.

Devi et al (1972) carried out a clinical review of eclampsia cases & found the incidence of eclampsia cases to be 1.4%, with 43% antepertum 43% intrapartum and 14% post partum eclampsia. They observed that 40% of the cases were from the rural areas, 80% of the cases were below 25 years of age and 75% patients were primigravida. The authors concluded that in this series, maternal mortality was 8.1% and perinatal mortality was 44.4%.

Weightmon et al (1974) studied perinatal morbidity and mortality associated with eclampsia. They found the total incidence of eclampsia to be 53/10000 deliveries, with a higher incidence among primigravidas and without any significant trend with the maternal age. In this series the perinatal mortality rate was 213 per thousand deliveries which was nine times higher than in control group. The authors observed main morbidity factors. to be intrauterine growth retardation and prematurity.

Page et al (1976) carried out a prospective study of hypertensive pregnant cases with respect to the impact of elevated blood pressure and/or proteinuria upon the pregnancy outcome. The authors observed that there was an increase in still birth rate, perinatal mortality, frequency of intrauterine growth retardation and neonatal morbidity in every category of the hypertensive disease of pregnancy except in gestational hypertension group.

Redman et al (1976) compared the effect of antihypertensive treatment upon the foetal outcome and they concluded that although perinatal mortality was lowerdd in the treated group, there was no beneficial effect on the birth weight and maturity of the babies in the treated group.

Redman et al (1977) carried out a controlled trial of methyldopa in cases of moderate hypertension in pregnancy and found that it was useful in the control of rise of blood pressure and an increasing daily dose of methyldopa was needed with advancement of pregnancy. The authors found the methyldopa to be a safe antihypertensive drug in terms of maternal & foetal side effects.

Jain et al (1978) studied the distribution of perinatal outcome in various groups of the hypertensive disorders of pregnancy (HDP). In this series the incidence of H.D.P. was 11.84% and the perinatal mortality (PNMR) rate was 106 per 1,000 deliveries. The observed a significant difference in the PNMR, in booked and unbooked cases, with PNMR to be 25.6 per 1,000 in booked cases and 394 per 1000 in unbooked cases ($p < 0.01$). The authors observed that 6.24% infants had birth weight less than 2 SD & 17.3% had birth weight less than 1 SD from the mean birth weight, for corresponding gestational age.

They further concluded that 17.44% the total perinatal deaths occured in the pre term babies and 95.88% of perinatal deaths occured in low birth weight babies. During this study they observed that there was direct co-relation between the severity of the PET and the perinatal loss and the PMR was 60, 172.7 and 378 per 1000 respectively in cases of mild PET, severe PET and eclampsia ($p < 0.01$). A similar effect of severity of essential hypertension on foetal prognosis was observed and superimposition of PET in cases of even mild hypertension adversely affected the prognosis.

Naeye et al (1978) in their study found the incidence of H.B.P. to be 3.2% and the perinatal mortality rate to be 57.9 per 1000 deliveries in comparison to 17.2 per 1000 deliveries in control group. They observed that out of all perinatal mortality, 42% were due to placental infarctions, 15% due to IUGR and 13% due to abruptio placente. Yoon et al (1979) studied the relationship betwee maternal hypertensive disease of pregnancy and the incidence of iddopathic respiratory distress syndrome. They observed that IRDS was lower in HDP (15.2% in comparison to normotensive group (29.9%) ($p < .001$) and the distribution of total cases of IRDS was 20% in mild PET, 13% in severe PET and 7.1% in eclampsia. They further concluded that the mortality rate of the infants without IRDS was significantly higher in the HDP mothers (22.6%) than mortality rate in infants of the non HDP mothers(16.5%).

Long et al (1980) observed that the prevalence of IUGR was 8.7% in preeclamptic patients in comparison to 8.6% prevalance in control group. and the prevalance of intrauterine growth retardation was most marked in early onset preeclampsia (18.7%). They also concluded that the perinatal death rate was higher in pregnancies with early onset preeclampsia.

Chin Chue et al (1981) evaluated the foetal outcome in hypertensive disorders of pregnancy. They found that the perinatal outcome was extremely poor in the study with the perinatal mortality to be 134 per 1000 births. In addition 22% of the infants were small for the date and 40% of the infants were born before term. They observed that the maximum perinatal mortality (81%) was in preeclamptic group. The worst foetal outcome was encountered in multiparous eclamptic women. The authors observed the incidence of caesarean section to be twice as common as in the control group and the incidence of low APGAR score was higher in the study group.

Rubin et al (1982) carried out a placebo controlled trial of atenolol in treatment of pregnancy associated hypertension. The authors found the similar incidence of intrauterine growth retardation, neonatal hypoglycaemia and hyperbilirubinaemia in both groups, although the occurance of respiratory distress syndrome was more in placebo group. They furthers observed that

the mean delivery time was 38±16 week in the placebo group and 39±1.0 weeks in the atenolol group.

Brazy S.et al (1982) studied the neonatal manifestations of severe maternal hypertension occuring before the thirty six weeks of pregnancy. In this series 39% of infants were small for age by 10th percentile and 96% babies were of birth weight lesser than the fiftieth percentile. The authors observed that the 50% of babies in study were delivered with a low APGAR score (Less than 5). They found the incidence of neonatal mortality to be same in both control and study group (7%). There were no case of still birth, or maternal death in this study group. They further observed that the infants of hypertensive mothers had a significant higher incidence of thrombocytopaemia, leukopenia, neutroperia, patent ductus arteriosus, hypotonia and gastrointestinal hypomotility.

Fidler et al (1983) carried out a randomized controlled comparative study of methyldopa and exprenalol in treatment of hypertension in pregnancy. They observed that the systolic blood pressure was better controlled in patients receiving exprenalol in comparison to methyldopa group although diastolic pressure was controlled in similar pattern. The authors found similar perinatal results in both groups.

Moore et al (1983) in their styd of hypertensive pregnant ladies, concentrated upon the early onset preeclampsia and concluded that the preeclampsia of early onset is responsible for higher perinatal morbidity, mortality and distinct maternal risk factors. In this series the authors identified certain risk factors for preeclamptic women i.e. a history of infertility, headaches, particularly migraine, preeclampsia in a previous pregnancy, or a raised serum alpha feroprotein concentration, although they could not associate other factors e.g. maternal age, a history of chronic hypertension or renal disease or excessive maternal weight gain with the occurance of preeclampsia.

Shbai et al (1984) tried to evaluate the effect of conservative management of severe preeclampisia of midtrimester onset upon the maternal and perinatal outcome. They found that there was a high incidence of maternal complications in form of abrptio placentae, eclampsia, coagulopathy, renal failure hypertensive encephalopathy, intracerebral haemorrhage and ruptured hepatic haematomas they also observed a high perinatal mortality (87%) in such cases.

MATERIAL & METHODS

MATERIAL AND METHODS

The present study was carried out in the Department of Obstetrics and Gynaecology, M.L.B.Medical College, Jhansi in active collaboration with the Department of Paediatrics and Department of Medicine over a period of 12 months from July 87 to June, 88. The case material for the present study was obtained from the mothers and the newborns delivered in M.L.B.Medical College Hospital, Jhansi.

The study comprised of 200 cases, out of which 50 served as control and 150 cases were labelled as Hypertensive cases.

The whole study group was classified into two broad groups.

1. Control group :-

This group comprised of normotensive antenatal cases without any obstetrical complication and who delivered normally.

2. Hypertensive group :-

This group comprised of the patients with pregnancy more than twenty eight weeks and in whom, the blood pressure was recorded more than 140/90 at two occasions; 24 hours apart, with or without proteinuria and/or convulsions.

Selection of controls

Fifty cases of normotensive pregnancies served as control cases. The criteria for selection was

1. These patients were normotensive i.e. blood pressure recording was less than 140/90 mm Hg. throughout their antenatal period.
2. There was no associated obstetrical complications in any control case.

Selection of study group

The study group comprised of one hundred and fiftey cases of hypertensive disease of pregnancy. The criteria for selecting the cases were:

1. These patients were having singelton pregnancy of more than 28 weeks duration, with blood pressure of more than 140/90 mm Hg, recorded at two occassions, 24 hours apart.
2. These hypertensive pregnant females were tested for proteinuria and the presence of proteinuria was defined as 1+ or more by albustic.
3. In eclampsia cases, associated convulsions were observed.

Classification of Hypertensive patients

The hypertensive patients were classified into four subgroups:-

(1) Pregnancy induced hypertension.

(2) Mild preeclamptic toxaemia.

(3) Severe preeclamptic toxaemia.

(4) Eclampsia.

Criteria for each group

Pregnancy induced hypertension (P/H)

Those cases were termed as P/H in whom the blood pressure recording was more than 140/90 mm Hg and there was no proteinuria.

Mild preeclamptic toxaemia (Mild PET)

In this group of HDP, the blood pressure recording was more than 140/90 mm Hg, but less than 160/110 mm Hg and with associated proteinuria.

Severe Preeclamptic toxaemia (Severe PET)

In this group the blood pressure recording was more than 160/110 mm Hg and proteinuria was also present in every case.

Eclampsia

In this group, patients had blood pressure recording more than 140/90 mm Hg. had proteinuria along with convulsions.

In this series we were not able to find any patient with essential hypertension or secondary hypertension with or without superimposition of toxaemia of pregnancy.

Oedema was not considered as a criteria for toxaemia as per different studies, since it has got no effect on the perinatal outcome.

Transient hypertension and proteinuria are common finding in labour and if such cases would have been included, a false impression of incidence and results will be obtained. So patients who had a raised blood pressure without proteinuria, only during labour were excluded from this study.

Patient were also considered unsuitable for the trial if they had any other major obstetrical problem such as diabetes, Rhesus immunization and multiple pregnancies.

The whole study group was divided into two broad groups, according to treatment given :-

1. Treated group :- included hypertensive patients who received antihypertensive treatment.
2. Untreated group :- included those hypertensive patients who could not receive any antihypertensive treatment because of very late detection, mainly due to improper antenatal case.

A detailed clinical history was taken and an extensive examination was done in each case.

HISTORY OF EXAMINATION

History of present pregnancy

1. Age
2. Socioeconomic status
3. Literacy status
4. Resident of rural or urban area

5. Period of amenorrhoea
6. Date of quickening
7. Antenatal care, received or not.
8. Blood pressure recording during antenatal period and any treatment given for it.

History of Past Illness

History of - hypertension
- renal disease

Family History

History of hypertension and diabetes in family.

Obstetrical History

A detailed account of the previous pregnancies was taken special emphasis on the following points.

1. Number of pregnancies & outcome of each pregnancy.
2. Any perinatal loss.
3. History of hypertension in any previous pregnancy.

Examination of mother

A through examination, general and systemic of mothers was carried out with special emphasis on the blood pressure recording, evidence of paller, oedema and weight gain.

Blood Pressure Recording

The blood pressure of each patient was recorded with the help of a mercurial manometer, which was standardised against a standard mercurial manometer frequently. The disappearance of the Kortakoff sounds (Phase V) was

taken as measurement of diastolic blood pressure as according to Rafferye and Ward 1969, phase V appears to corelate better with diastolic blood pressure than the muffling of sounds.

For Abdominal Examination

1. The fundal height was noted and corelated with the period of amenorrhoea.
2. The presentation as well as the degree of engagement was looked for.
3. The foetal heart was auscultated, noting its rate, rhythm and intensity.
4. If the patient was in labour, uterine contractions were felt and the duration, intensity and number in 10 minutes, alongwith relaxation was noted.

For Vaginam Examination

It was done if patient was in labour or induction of labour had to be done.

1. The pelvis was assessed to exclude cephalopelvic disproportion.
2. The condition of the cervix was noted and bishop's scoring done.

Investigations

Blood - Haemoglobin
Total leukocyte count
Differential leukocyte count
Blood urea

Serum creatinine

Blood sugar

Serum cholesterol

Urine examination for - Albumin

Sugar

Microscopic for R.B.Cos.

Pus cells count.

Fundoscopic Examination - For retinal changes

Electro-Cardiography for cardiac changes

Selection of cases for treatment

Hypertensive pregnant patients were spotted in the antenatal clinics and further line of treatment was dependent on the maturity of the foetus.

In cases with gestational age less than 37 weeks, conservative management was attempted to prolong the pregnancy until foetal lung maturity was achieved or until onset of either maternal or fetal complications occured.

So far as possible treatment was carried out as an outpatient basis and patients were admitted to hospital only if the blood pressure was difficult to control or if they developed any complication.

Drugs

Sedative groups - Diazepam (Calmpose) was mainly used

Dose ranged from 5 mg to 20 mg/day

oral/parentral

Diuretic groups - Lasix (Frusemide) was used
Tab. or injection
Dose range 40-80 mg/day

Hypotensive groups -

-Tab. Methyldopa was used in all cases
25 mg tab. orally given.
for maximum dose range 0.5 to 4 g/day.
-Cap. Calcigaard (5 mg) used as
adjuvant in few cases.

Obstetrical Management

The conservative treatment had to be discontinued in both treated and untreated group with the onset of any maternal and/or foetal complications.

Our main aim was to shorten the total duration of labour for safety of mother and/or baby and it was acheived by any of the methods of induction and if they failed by doing caesarean section.

Examination of New born

Just after birth, the baby was examined for APGAR score index, birth weight and for evidence of any life treatening congenital anomalies.

Subsequently a thorough general and systemic examination was done during the hospital stay. The babies cry colour activity, posture, gestational age and anthropometric measurement were noted in each case. Special epphasis was given to observe the presence of neonatal

sepsis (superficial and deep), jaundice, bleeding diethesis and neonatal systemic disease. The cause of neonatal death was also ascertained in each case.

Gestational Age Assessment

The gestational age of the baby was determined by examination of the various morphological criteria - (From Neldon; textbook of Paediatrics page 365).

Gestational age

1. Nipple (size)	Not palpable (< 34 weeks)	3-4 mm (34-36 weeks)	4-10 mm (at term)
2. Planter creases (Extent)	Ant. 1/3rd (< 36 weeks)	Ant 2/3rd (37-38 weeks)	Criss cross creases on sole (40 weeks)
3. Ear Cartilage	Not formed (< 36 weeks)	formed (36-40 weeks)	
4. Scalp Hair	Short fuzzy (< 37 weeks)	Long coarse (37 weeks)	
5. External genitalia	Male Undescended testis (< 37 weeks)	Decended testis (at term)	
	Female Labia majora donot cover the minora (< 37 weeks)	Labia majora cover the L.minora (at term)	

Perinatal outcome

Perinatal period is the period which extends from the 28th weeks of gestation to the 7th day of neonatal life.

Perinatal mortality includes still births and early neonatal deaths.

Gestational age groups - According to the period of gestation the new born baby is classified as

1. Premature or Preterm - A premature infant is defined as a baby with gestational age of less than 37 weeks.
2. Term are those babies having gestational age between 37-41 weeks.
3. Post term babies babies having gestational age of 42 weeks or more are classified as post term or postmature babies.

Groups according to birth weight

Low birth weight (LBW)- Babies with a birth weight of 2.5 kg or less irrespective of the period of gestation are called low birth weight babies. These include term, preterm and post term babies.

Small for date (SGA) - babies with a birth weight of less than 10th percentile below the mean birth weight for that gestational age are called small for date babies.

Appropriate for gestational age babies with a birth weight between 10th to 90th percentile or between 2 standard deviations of the mean birth weight for the gestational age are known as appropriate for gestational age babies.

Large for gestational age - Babies with a birth weight of more than 90th percentile or 2 standard deviation above the mean for the gestational age are known as large for gestational age babies.

By combining classification of the babies on the basis of gestational age alone and with gestational age and birth weight the new born population was divided into the following 9 groups.

1. Preterm : I. Small for date SFD
   II. Appropriate for gestational age AGA
   III. Large for gestational age LGA
2. Term : I. Small for date
   II. Appropriate for gestational age
   III. Large for gestational age.
3. Post term: I. Small for date
   II. Appropriate for gestational age
   III. Large for gestational age

In this series intrauterine growth charts drawn on the basis of a study in the all India Institute of Medical Sciences (from Meharban Singh book " Care of the New born ") were the guidelines to determine the extent of intrauterine growth retardation.

Anthropometric measurements :

- Weight
- Head circumference - was measured at occipitofrontal level.
- Chest circumference - was measured at level of nipples.
- Length was measured from vertex to heel.

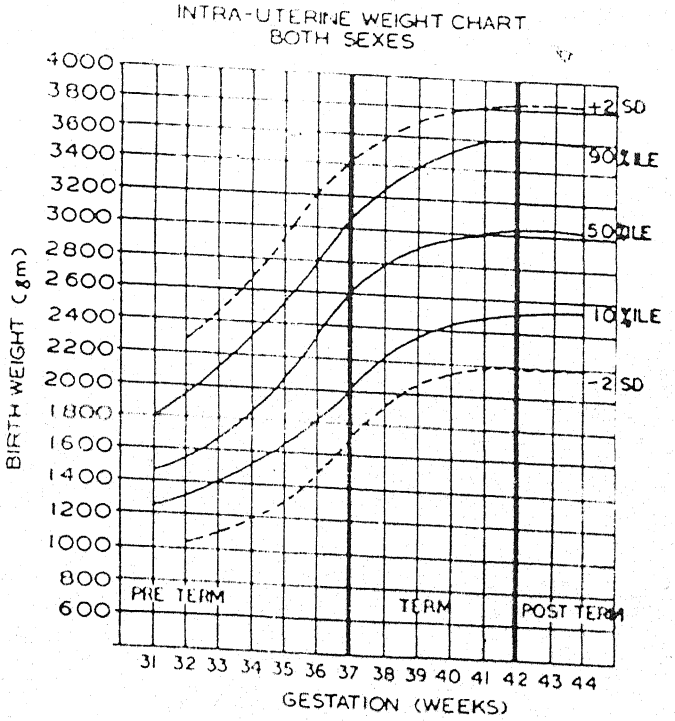


Figure 9.1 Intrauterine weight charts (AIIMS)

OBSERVATIONS

OBSERVATIONS

The present study was carried out in the Department of Obstetrics and Gynaecology over a period of twelve months from July 87 to June 88. Our observations are tabulated as follows:-

Table I

Showing the percentage of hypertensive disorders of pregnancy (HDP) in general population.

Total deliveries during study period	: 1218
Number of HDP cases	: 150
Percentage of HDP	: 12.3%

It is evident from the table I that the incidence of hypertensive disorders of pregnancy was 12.3% in this series.

Table II

Showing the study group.

	Group	No.of cases
I	Control	50
II	Study	150
	A. Pregnancy induced hypertension	48
	B. Preeclamptic toxaemia	
	Mild	44
	Severe	33
	C. Eclampsia	25

Table II shows the number of cases in study and their distribution in different groups. In this study the number of control cases was 50 while there were 150 cases of hypertensive disorders of pregnancy. Out of these 150 cases 48 cases were of pregnancy induced hypertension, 44 cases of mild preeclampsia 33 cases of severe preeclampsia and 25 cases of eclampsia.

Table III

Showing the distribution of hypertensive pregnancies, according to the age of the patient.

	Group I $\leq$ 20 yrs.	Group II 20-30 yrs.	Group III $\geq$ 30 yrs.
Control (A)	18% (9)	44% (22)	38% (19)
H.D.P. (B)	24.6% (37)	40% (60)	35.4% (53)

I A	Vs	B	P > .05
II A	Vs	B	
III A	Vs	B	

It is evident from the table III that there is no significant difference in distribution of hypertensive pregnancy patients in different age group in comparison to control cases. A majority of cases, 62% in control group and 65% cases in the study group were below the thirty years of age.

Table IV

Showing the distribution of hypertensive pregnant cases according to educational status.

	Group I Illiterate	Group II Schooling upto 8years	Group III Higher education
Control (A)	20% (10)	50% (25)	30% (15)
H.D.P. (B)	64% (96)	20% (30)	16% (24)

Group I A Vs B
II A Vs B
III A Vs B
$p < .01$

Table IV illustrates a higher percentage of HDP in illeterate female (64%) in comparison to control (20%), while in educated group, the incidence of HDP is lower. 80% of control group were educated, out of these 50% patients received primary education, while in HDP group only 36% were educated.

Table V

Showing the distribution of hypertensive pregnant cases according to socio-economic status (monthly income).

	Group I ₹ 500	Group II 500-₹ 1000	Group III 1000+
Control (A)	20% (10)	60% (30)	20% (10)
HDP (B)	50% (75)	35% (53)	15% (22)

Group I A Vs B
II A Vs B
III A Vs B
$p < .01$

It is evident from table V that the lower socioeconomic class females are more prone to develop HDP in comparison to middle and higher socioeconomical calasses. 50% of the study group (HDP) were from the lower socioeconomic group, while 80% of the control group belonged to middle or higher socioeconomic group.

Table VI

Showing the distribution of hypertensive patients according to parity.

	Group I (Primi)	Group II (Multi).
Group (A)	30% (15)	70% (35)
HDP (B)	64% (97)	36% (53)

Group I A Vs B } p < .01
II A Vs B }

In HDP I Vs II p < .01

It is evident from the table VI that in the control cases, 70% were multigravida, while 30% were primigravidae. On the other hand in the HDP group, proportion of the primigravida was more i.e. 40% were primigravida & 36% were multigravida.

Table VII

Showing distribution of perinatal deaths according to Parity.

	Group I (Primi)	Group II (Multi)
Control (A)	0	0
HDP (B)	35% (33)	23% (12)

In HDP I Vs I p < .05

Table VII shows that the perinatal mortality was significantly higher in primigravida (35%) in comparison to multigravida (23%).

Table VIII

Showing the difference in perinatal loss according to the antenatal care.

	Group I No antenatal care	Group II Poor to fair antenatal care
Control (A)	n = 26	n = 24
% of perinatal loss	0	0
HDP (B)	n = 90	n = 60
% of perinatal loss	41.6% (37)	12.5% (8)

Group I A Vs B } p < .01
II A Vs B }

In HDP group I Vs II p < .01

It is clear from the table VIII that the antenatal care has improved the perinatal survival in the hypertensive group. The perinatal loss was only 12.5% in booked cases while it was as high as 41.6% in unbooked ladies.

Table IX

Showing the difference in perinatal loss according to haemoglobulin level (g%).

	Group I <7.5	Group II 7.5-10	Group III >10
Control (A)	n = 20	n = 25	n = 5
% of perinatal loss	0	0	0
HDP (B)	n = 90	n = 60	n = 50
% of perinatal loss	41.6%(37)	12.5%(8)	0

Group I A Vs B $p < .01$

II A Vs B $p < .05$

In HDP group I Vs II $p < .01$

It is clear from the table IX that there was a significant increase in the perinatal loss in the anaemic patients. It was as high an 41.6% in severely anaemic patient while these was no perinatal death in group III i.e. patients with a satisfactory level of haemoglobin.

Table X

Showing the difference in neonatal loss according to birth weight (kg.).

	Group I < 2.5 kg	Group II ≥ 2.5 kg
Control (A)		n = 50
% of neonatal loss	0	0
	n = 30	n = 91
HDP (B)		
% of neonatal loss	61.5% (16)	0

Group I A Vs B $p < .01$

II test not applied

In HDP I Vs II $p < .01$

Table X is showing that there was no low birth weight baby in the control group, while there were 30 such babies in HDP group. It is also evident that there babies are not higher risk, as there was 61.5% neonatal loss in the low birth weight babies while there was no neonatal death in the new borns, delivered with adequate birth weight.

Table XI

Showing the difference in neonatal loss according to gestational age.

	Group I < 37 weeks	Group II ≥ 37 weeks
		n = 50
Control (A) % of neonatal loss	0	0
	n = 28	n = 93
HDP (B) % of neonatal loss	28.6% (8)	8.5% (8)

Group I	A	Vs	B	p < .01
II	A	Vs	B	p < .05
In HDP	I	Vs	II	p < .01

It is clear from the table XI that there was no premature delivery in the control group, while there were 23.2% premature births in the HDP group. There was a significant increase in the neonatal deaths in such prematurely delivered babies (28.6%) as compared to neonatal loss in the term babies (8.5%) of HDP patients.

Table XII

Showing the difference in neonatal loss according to the intrauterine growth.

	Group I IUGR	Group II AGA
		n = 50
Control (A) % of neonatal loss	0	0
	n = 21	n = 100
HDP (B) % of neonatal loss	66.6% (14)	20% (2)

IUGR = Intrauterine growth retardation

AGA = Average for gestational age.

Group I A Vs B
II A Vs B } p ∠ .01

In HDP I Vs II p ∠.01

Table XII shows that there was no small for date baby in the control group, while 17% babies of hypertensive pregnant ladies were small for date. There was also a significant higher neonatal mortality in growth retard babies (66.6%) in comparison to 20% neonatal deaths in average for gestational age babies in HDP group.

Table XIII

Showing the perinatal outcome in hypertensive pregant ladies in comparison to control gases.

Perinatal outcome	HDP group	Control group	p A Vs B
Perinatal mortality	30% (45)	0	∠ .01
Still births	19% (29)	0	∠ .01
Neonatal deaths	13.2%(16)	0	∠ .01
Term & average for gestational age	66.9%(81)	100%	∠.05
Premature babies	23% (28)	0	∠ .01
Small for date babies	17.3%(21)	0	∠ .01
Term & SFD	9.9%(12)	0	∠ .05
Preterm & SFD	7.4%(9)	0	∠ .05

Table XIII represents a poor perinatal outcome in hypertensive pregnant ladies in comparison to hormotensive pregnant ladies in comparison to normotensive control

group. There was a 30% perinatal mortality in HDP group, while there was no perinatal death in the control group. In this perinatal salvage, 19% cases were still births and 13.2% babies expired in the neonatal period. The occurance of term & average for gestational age babies was 66.9% in HDP & 100% in control group. There was no premature or small for date baby in control group, while there were 13% premature babies and 17.3% small for date babies in HDP group. Among small for date babies, 99% were delivered at term. While rest were premature babies.

Table XIV

Showing the perinatal outcome in different groups of study.

Group	Perinatal mortality	Still births	Neonatal deaths	Pre-mature babies	Small for date
Control	0	0	0	0	0
P/H	8.3% (4)	6.3% (3)	2.2% (1)	15.5%(7)	4.4%(2)
Mild pet	24.1% (11)	13.8%(6)	13.1% (5)	22.2%(8)	11.1%(4)
Severe pet	31.8% (10)	27.2%(8)	8.0% (2)	28.0%(7)	28.0%(7)
Eclampsia	82.3% (20)	47.0%(12)	61.5% (8)	46.2%(6)	61.5%(8)

Perinatal mortality

P/H $< .05$
Mild PET, Severe PET, Eclampsia $p < .01$

Still births

P/H, Mild pet $p < .05$
Severe PET, Eclampsia $p < .01$

Neonatal deaths

P/H, Mild PET $p < .05$
Severe PET, Eclampsia $p < .01$

Premature babies

p/H $< .05$
Mild PET, Severe PET, Eclampsia $p < .01$

Small for date babies

p/H $> .05$
Mild PET, Severe PET, Eclampsia $p < .01$

Table XIV illustrates the difference in perinatal outcome in different grades of toxaemia of pregnancy. It shows that the perinatal mortality was maximum in eclampsia groups (82.3%), while it was 31.8% in severe preeclampsia, 24.1% in mild preeclampsia and only 8.3% in pregnancy, induced hypertension cases. In P/H group, 6.3% were still birth and 2.2% of live births expired in neonatal period. There were 13.8% still birth & 13.2% neonatal deaths in mild preeclampsia group. In severe preeclampsia group, there was a higher incidence of still births (27.2%) as comparison to neonatal deaths (8%). Although in eclampsia group a 61.5% neonatal salvage was seen in comparison to 47% still births. There was a rising trend of premature births with the severity of toxaemia. The percentage of premature babies was 15.5% in pregnancy induced hypertension. 27.2% in mild preeclampsia, 28% in severe preeclampsia and 46.2% in eblampsia. Similar distribution of small for date babies was noted & the percentage of such babies was 4.4% in P/H, 11.1% in mild PET, 28% in severe PET and 61.5% in eclampsia.

Table XV

Showing the comparison of perinatal outcome in treated and untreated hypertensive pregnant ladies.

	Perinatal deaths	Still births	Neonatal deaths	Premature	Small for date	Full term AEG
Group (A) 112	35.7%(40)	23%(26)	15.1%(13)	14%(12)	11.6%(10)	42%(47)
Treated(B) 38	13.1%(5)	7.8%(3)	8.6%(3)	45.6%(16)	31.4%(11)	63.1% (24)
P A Vs B	L.01	L.01	L.01	L.01	L.01	L.05

In table XIV the perinatal outcome in treated cases of HDP was compared with that in the untreated group. The perinatal deaths was strikingly higher in untreated group (35.7%) in comparison to treated group (13.1%).

Out of these perinatal deaths, the number of still birth (23%)was higherthan neonatal (15.1%) salvage in untreated group, while there was an equal distribution of both types in treated group.

The premature deliveries were significantly higher in treated (45.6%) group as comparison to untreated group (14%). Similarly a higher number of small for date babies were born in treated group (29%) as comparison to 8.9% in untreated group. The incidence of full term and average for date babies was 63.1% in treated HDP group & 42% in untreated group.

Table XVI

Showing the perinatal outcome in treated and untreated cases in different subgroups of HDP.

Group	Perinatal mortality	Still births	Neonatal deaths	Premature babies	Small for date babies
Pregnancy induced hypertension					
treated(A) (18)	0	0	0	11.1%(2)	5.6%(1)
Untreated(B) (30)	13.2%(4)	10%(3)	3.3%(1)	16.7%(5)	3.3%(1)
p value A Vs B	$<.05$	$>.05$	$>.05$	$>.05$	$>.05$
Mild pre-eclamptic toxaemia					
treated(A) (9)	0	0	0	22.2%(2)	0
Untreated(B) (35)	31.4%(11)	17.1%(6)	14.3%(5)	17.6%(6)	11.4%(4)
p value A Vs B	$<.01$	$<.05$	$<.05$	$>.05$	$<.05$
Severe pre-eclamptic toxaemia					
treated(A) (11)	27.3%(3)	18.2%(2)	0	18.2%(2)	9.9%(1)
Untreated(B) (12)	31.8%(7)	27.3%(6)	9.9%(2)	22.7%(5)	27.3%(6)
p value A Vs B	$>.05$	$>.05$	$<.05$	$>.05$	$<.05$

Table XVI represents a good prognostic effect of the antihypertensive treatment upon the perinatal out come. This positive effect was especially marked in the

milder forms of hypertension. There was no perinatal death in treated p/H & mild PET cases, while it was 13.3% in untreated p/H cases and as high as 31.4% in untreated mild PET cases. On the other hand there was no statistically significant difference of perinatal mortality in treated (27.3%) & untreated group (31.8%) of severe PET.

In all the three groups, the percentage of intrauterine deaths was more in comparison to neonatal deaths.

It is also interesting to note that the incidence of premature and small for date babies was not significantly different in treated and untreated group in all grades of toxaemia.

Table XVII

Showing the comparison of the incidence of caesarean section in the hypertensive group to those in the general obstetrics population.

		Group I Hypertensive group n=150	Group II General obstetric population n=1068
Ceasarean section	No.	59	195
	%	39%	17.3%

I Vs II p< .01

It is evident from Table XVII that a higher percentage of hypertensive pregnant patients had undergone operative delivery (39%) in comparison to general obstetric population (17.3%).

Table XVIII

Showing the distribution of perinatal loss according to mode of delivery.

Mode of delivery		Number	Perinatal mortality
Normal vaginal	A	79	25
Forceps	B	12	3
Caesarean section	C	59	17

Group A Vs B, B Vs C, A Vs C } p > .05

Table XVII represents that there was no effect of mode of delivery upon the perinatal mortality in the study group In this series 60.6% were vaginal deliveries while rest were delivered by caesarean section.

Table XIX

Showing the effect of caesarean section upon the perinatal outcome in treated and untreated groups of HDP.

	Group I Treated HDP cases	Group II Untreated HDP cases
No.of caesarean deliveries	22	38
Perinatal mortality	0	17

group I Vs II p < .01

It is clear from the table XIX that operative intervention has improved the perinatal servival in treated group.

Table XX

Showing the comparison of the incidence of low APGAR score (at ≤ 6 at 1 minute) in the hypertensive group to those in the control group.

	Group I Hypertensive group (150)	Group II Control group (50)
No.	30	1
Percentage	20%	2%

Group I Vs II $p < .01$

Table XX represents that a significant higher number of babies with low birth APGAR were born in hypertensive group (20%) in comparison to control group (2%).

Table XXI

Showing the perinatal mortality ratio in different group of HDP.

Group	PMR/1000 births
Pregnancy induced hypertension	83.3
Mild Preeclamptic toxaemia	250
Severe preeclamptic toxaemia	303
Eclampsia	800
Control	0

It is clear from the table XXI that the perinatal mortality ratio was 83.3% in p/H, 250 in mild PET, 303 in severe PET and 800 in eclampsia group, while there was no perinatal salvage in the control group. The difference in perinatal mortality ratio of all these groups was statistically significant.

Table XXII

Showing causes of maternal mortality in hypertensive disorders of pregnancy (all were eclampsia cases).

Causes of death	No. of cases
Shock	2
Pulmonary oedema	1
Hepatic coma	1

Maternal mortality was 16% in eclampsia Table XXII showing that there were four maternal deaths in study group. All these were eclampsia patients. Out of there fore two patients died as a result of shock, while pulmonary oedema and Hepatic coma was the cause of death in two patients.

Table XXIII

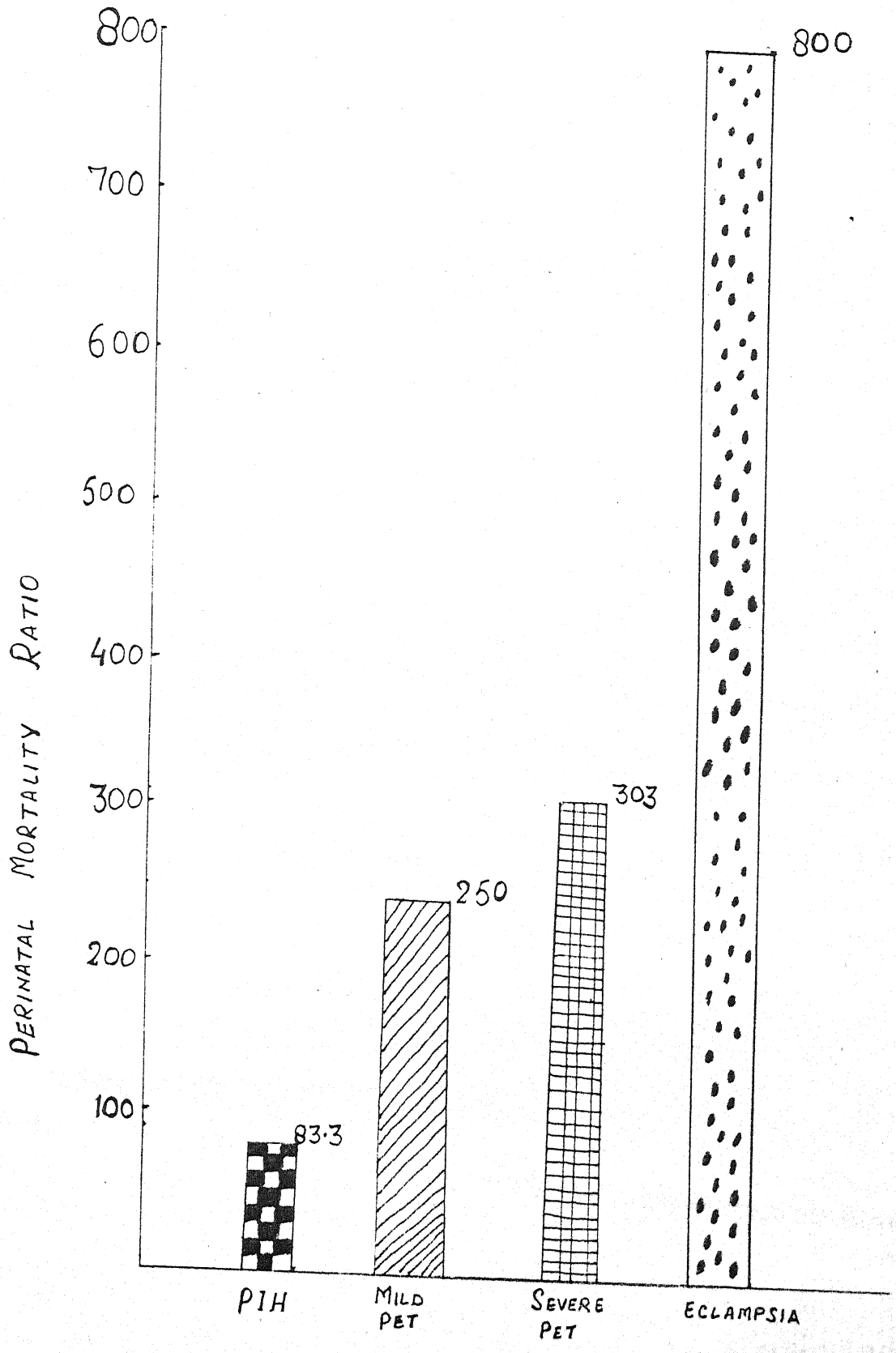
Showing the causes of perinatal deaths in hypertensive pregnant females.

Still births (29)		Neonatal deaths (16)	
Macerated still births (Placental insufficiency)	15	Prematurity (Suspected RDS)	8
Fresh still births	7	Asphyxia	4
Obstructed labours	6	Meconium aspiration syndrome	2
Congenital anomaly	1	Septaecemia	2

It is evident from table XXIII that the still births (29) consituted a higher proportion of the perinatal

salvage as comparison to neonatal deaths (16). The major bulk of the still birth group was of macerated babies (15). There were 7 fresh still births 6 babies died in utero because of obstructed labour and congenital anomaly was responsible for intrauterine death in only one case. The most common cause of neonatal death was prematurity and suspected respiratory distress syndrome (8). Asphyxia was the cause of death in four cases. There were two deaths due to meconium aspiration syndrome and another two succumbed to septicaecemia.

Diagram showing the Perinatal mortality ratio per 1,000 live birth in study graph



STUDY GRAPH

DISCUSSION

D I S C U S S I O N

Incidence of HDP

The present study was carried out in the department of Obstetrics and Gynaecology over a period of about one year. In this study there were 150 cases of hypertensive disorders of pregnancy (HDP). Out of 1218 total deliveries. Thus the percentage of HDP was 12.3% in this series. The incidence reported by other authors, is as follows -

Table XXIV

Authors	Year	Incidence of HDP
Western Countries		
Dewson	1942	4.33%
Hamilton	1949	5.8%
Nondanstrohl	1951	5.9%
Naye	1978	3.2%
India		
Jain	1982	11.84%

The high incidence of hypertensive disorders of pregnancy in our series is partly due to the referal nature of this hospital.

The maximum number of cases were of pre-eclampsia, while the incidence of eclampsia cases was 2.0%. Devi (1972) has reported this incidence to be 1.4%.

Age Factor

No significant difference was found in the mean age group of different types of HDP in our study. Similar results were reported by Jain (1978), Weightman (1978) and Brazy (1982). However Devi (1972) reported that 80% of the eclampsia cases were below 25 years of age.

Parity

Our study also confirmed the predisposition of primigravida for toxaemia. The perinatal mortality was higher in primigravidae (35%) in comparison to multigravida (23%). Lin (1983) however reported a higher perinatal mortality in multigravida.

Antenatal care

77% of the total perinatal deaths in this series occured in the unbooked patients. Jain (1978) and Jain (1983) also suggested the importance of antenatal care for the improvement of the perinatal outcome, as in their series also, about 80% of the perinatal deaths occured in unbooked cases.

Anaemia

There was a direct relation between the haemoglobin status and the occurance of HDP in our study. Besides this, the anaemic patients with HDP lost their babies more frequently. This result coincides with the findings of Jain (1983).

Low birth weight

We found a significant increase in the incidence of low birth weight babies in hypertensive group and interestingly all neonatal deaths were from the low birth weight group. Jain (1983) also observed low birth weight to be a risk factor for neonates of hypertensive females.

Premature births

There was a high incidence of premature babies in H.P group (23.2%) as compared to none in the control group. There was a increased incidence of neonatal deaths in those premature babies (28.6%) as compared to term babies (8.5%). Devi (1972) and Jain (1986) also reported similar results. Lin (1981) reported 30% premature births similar population.

IUGR

In this study the percentage of intrauterine growth retarded babies was 17% in study group. This incidence is very high as compared to the results of Jain (1978) i.e. 6.24%. Martines Tupper (1979) and Brazy (1982) have also reported a strikingly high incidence 56.5% and 29% respectively. This high incidence can be explained by a more conservative approach in obstetrical management in the later studies. We also observed that these babies are more prone for neonatal complications.

Perinatal mortality

In our series, the total perinatal mortality was 30%. Meuwelier (1948) reported a foetal wastage of 26.9%, while de-Rezende (1951) found it to be 20%. Lin (1981) reported this to be 31.4%. On the other hand Eastman (1950) observed 7.4% perinatal mortality and Gibson (1950) reported it to be 11.7%. Such a high incidence of perinatal mortality in our series especially in eclampsia cases (82.3%) can be explained by the fact that the eclampsia patients were brought in a very late stage for any treatment to be effective for the betterment of the baby. Furthermore the sedative drugs given to the mother may have contributed to the perinatal deaths as the sedatives cross the placenta and depress the foetal respiration which is already in jeopeady.

Effect of proteinuria

The perinatal mortality was significantly higher in nonproteinuric hypertensive patients (8.3%) as compared to control group (Nil) but overall perinatal outcome became poor with the further rise in blood pressure appearance of proteinuria and/or convulsions. Similar results were obtained by Nelstrop (1976) and Jain (1986) who observed that the level of blood pressure was the deciding factor in the perinatal outcome of the toxaemia although addition of proteinuria usually proved detrimental

Page and Christiensen (1976) and Friedman (1971 however found that the foetal salvage in women with gestational hypertension was not different from normotensive

pregnant ladies. They stressed the importance of proteinuria for the perinatal outcome.

Effect of treatment

There was a definite improvement in the perinatal survival in the treated group (P.N.M. 13.1%) in comparison to the untreated group (PNM 35%), despite the fact that the number of premature and IUGR births were higher in the former group. This was because of increased pregnancy of premature induction of labour in such cases, either to save mother and/or foetal complications. Smith and Bullein (1966) reported as low as 9.3% perinatal mortality in a similar population, similarly treated with methyldopa. Townsend (1958) reported the perinatal mortality in such cases to be 16%. Redman (1976) in this study found improvement in perinatal survival in treated group (with methyldopa) but there was no effect of the treatment on the birth weight and maturity of the viable infants. Leather (1968) also observed a positive effect of antihypertensive treatment upon the perinatal outcome.

The nonproteinuric hypertensive pregnant ladies responded better to antihypertensive treatment as compared to the proteinuric group. In severe preeclaptic group. There was no effect of treatment upon the foetal outcome. Our results coincides with the observations of MacGillivary (1963), Dixon.

Mode of delivery

The incidence of operative delivery in HDP cases (39%) was very high or compared to the general obstetric population (17.3%). Gibson (1950), Lin (1981) and Brazy (1982) has also reported similar results.

There was no significant effect of mode of delivery upon the perinatal outcome in hypertensive pregnancies. Out of total deliveries, 60.6% were vaginal and rest were caesarean sections.

It is interesting to note that in the treated group, there was no perinatal death in cases, delivered by caesarean section while there was a significant number of perinatal deaths in similar population of untreated group. Richton (1968), Leon (1968) and Villesen & Slabbers (1970) observed a better perinatal outcome in thr caesarean section deliveries.

APGAR Score

Birth asphyxia as measured by APGAR score was seen more often in HDP cases (20%) as compared to controls (2%) Lin & Brazy (1982) have also reported similar results.

Perinatal mortality ratio

We observed a marked rise in the perinatal deaths per 1,000 live births, in HDP cases. The highest perinatal mortality ratio was noted in Eclampsia patients

(800 per 1000 live births). These results coincide with the findings of Jain (1982).

Maternal Mortality

There was 3.7% maternal mortality in all cases of HDP, while it was 16% in eclampsia cases. All four maternal deaths were from the eclampsia group. Maternal mortality as reported in the literature is variable, thus it was M

Memon 2.2% (1961)	Crichton 8.4% (1968)
Upadhya 3.0%(1964)	Lopez & Letera 10.3(1967)
Strish & Mensif 7% (1968)	Devi 10.4% (1972)
Villers & Slabberr 8.2%(1970)	

Perinatal mortality

A significant proportion of perinatal deaths in our series was caused by an unfavourable, intrauterine environment. It is interesting to note that more than half of the still births in our series were macerated. The neonatal deaths were mainly attributed to complications of prematurity. The duration of hospitalization of HDP infants was longer than in control group. Almost similar results were reported by Dienna (1941). Jain(1983) & Jain (1987).

CONCLUSION & SUMMARY

SUMMARY & CONCLUSIONS

1. 150 cases of toxaemia of pregnancy were studied over period of 1 year from July 87 to June 88.
2. The perinatal outcome in terms of morbidity and mortality was observed in treated and untreated cases.
3. The overall perinatal mortality ratio was 300 per 1000 live births in hypertensive cases (Nil in control).
4. There were 45 perinatal deaths in study group & more than half of there were still births.
5. 17.3% infants were small for date (∠25d) while 23% babies born before term and 20% newborns were low birth weight (∠2.5 kg).
6. There was a definite increase in the perinatal mortality with the severity of the blood pressure and with the apperance of proteinuria and convulsions. The perinatal mortality was 8.3% in pregnancy induced hypertension (the mildest forms) while it was as high as 82.3% in eclampsia. It was 24.1% in mild preeclamptic PET toxaemic and 31.8% in severe PET cases.
7. The major risk factors increasing the perinatal mortality rate statistically, were the lack of antenatal care, Haemoglobin less than 8.5 g%. gestation less than 37 weeks, birth weight less than 2.5 kg and the presence of intrauterine growth retardation.
8. 60.4% cases were delivered via vaginal route, while rest were caesarean deliveries. There was no effect

of the mode of delivery upon the perinatal outcome.

9. The overall maternal mortality was 3.7%. All four deaths were from eclampsia group, thus constituting 16% incidence of maternal mortality in eclampsia group.

10. There was a definite improvement in the perinatal survival in treated hypertensive pregnant ladies (PNM 13.1%) as compared to untreated group (PNM 35.7%).

11. The incidence of prematurity and small for date babies was strikingly high in the treated group. i.e. 43.6% and 31.4% in comparison to untreated group with 14% premature and 11.6% small for date babies.

12. The positive prognostic effect of antihypertensive treatment was maximum in the mild form of hypertension.

13. There were a higher number of babies born with a low APGAR in study group (20%).

14. Intrauterine death was the more common cause of perinatal salvage in comparison to neonatal deaths.

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APPENDIX

WORKING PROFORMA

" PERINATAL OUTCOME IN CASES OF HYPERTENSION PREECLAMPTIC TOXAEMIA AND TOXAEMIA OF PREGNANCY TREATED AND UNTREATED CASES".

Serial No. Employment - Husband

Name - Wife

Address Monthly Income

Education - Husband

- Wife

Age

Admission Date Time Indication Ward/Bed

C/O Parity

	Physically	Neurologically
Damaged infants		
Abortions		
Premature		
Still birth /IUD		
Major congenital anomaly		
Neonatal death		
Birth weight of babies		

H/O APH. Toxaemia Forceps LSCS

H/O Any medical disorder

PRESENT PREGNANCY

Nutrition

No.of antenatal units Booked/unbooked cases

L.M.P. E.D.D.

IUGR/IUD

Gestational age of foetus Twins Hydramnios

Fundal height APH

Investigations :

Blood Haemoglobin
Blood Urea
Blood Sugar
Serum creatinine
Urine albumen
M/E
ECG
Fundus

Contd..2

Labour

FHR abnormality

Meconium staining of liquer

Duration

Spontaneous or induced

N./Forceps/LSCS

Results.

B.P. with details of treatment & investigation

Pregregnant level — Essential

Normotensive — Hypertensive

See

During Pregnancy (sodium, urine albumin)

Date	B.P.+Treatment	Date	B.P.+Treatment

At admission

Date	B.P.+ Treatment	Date	B.P.+Treatment

Examination of Baby

1. APGAR at 1 minute	Clinical colour	0 Blue/Pale	1 Bodypink limbs blue	2 All over pink
at 5 minutes	Heart rate	Absent	Less than 100/mm	7 100 min.
	Response to catheter in nostril	No response	Grimace	Cough or sneeth
	Activity	Limp	Some flexon of limbs	Active movement
	Respiratory efforts	Absent	Slow irregular	Good crying

Contd..3

2.Congenital anomaly

3.Birth weight

4.Length

5.Head circumference

6.Chest circumference

7.Maturity assessment

Extranal sign	Score 0	1	2	3	4
Edema	of hands & feet pitting over tibia	Only pitting over tibia	no		
Skin colour	Dark red	Uniformly pink	Variable pink	Pale	
Skin texture	very thin	thin & smooth	Superficial peeling	Slight thick superficial peeling	Thick parchment like
Skin opacity (Trunk)	Veins seen clearly	Veins &Tributeries seen	few large vessels	not clear	No vessel seen
Lanugo (over back)	No	Abundent & long thick	Thin	Occasionaly	more than half of back is devoid
Planter creases	No	Faint red over more than antirior	Definite red over more ant. half	Indentation over more ant.3rd	Deep definite over more than ant.3rd
Nipple	No areola barely visible	Areola smooth ⩽0.75cm	Areola stippled flat ⩽.75cm	Areola Stippled Edge raised ⩾0.75cm.	
Breast size	No	⩽.05cm	.5-1cm	⩾ 1cm.	
Ear	Pinna flat Shapless	Incurving of part of edge	Incurving whole edge.	well defined incurving	
Ear firmness	Soft,no recoil	Slow recoil	ready recoil	firm	

Genitalia Male	Neighter testis scrotum	At least one testis	Both
Female	Labia majora widely seperate	L.Majora almost covering L.Minora	L.Majora completely covering L.minora

Total Score

8.Maturity

Follow up

1. General appearance -

Date

Cry physical activity						
2. Skin	Oedema					
Vernix	Cynosis	Jeterus	Paller	Nails	Mongal ion aperta	Netting

3. Head: Caput Succedenmum -Cephalaematoma -Mouleling
Size of Anterior -Craniotabes
Fontanelle

4.Face and Neck Date

5.Systemic Lung
Heart
Abdomen
Genitalia
Excretory
CNS

6.Treatment.